

1986

Abdominal Binding and Counterpulsation in Cardiopulmonary Resuscitation

Charles F. Babbs

Purdue University, babbs@purdue.edu

William E. Blevins

Follow this and additional works at: <http://docs.lib.purdue.edu/bmepubs>



Part of the [Biomedical Engineering and Bioengineering Commons](#)

Recommended Citation

Babbs, Charles F. and Blevins, William E., "Abdominal Binding and Counterpulsation in Cardiopulmonary Resuscitation" (1986).
Weldon School of Biomedical Engineering Faculty Publications. Paper 112.
<http://docs.lib.purdue.edu/bmepubs/112>

This document has been made available through Purdue e-Pubs, a service of the Purdue University Libraries. Please contact epubs@purdue.edu for additional information.

Abdominal Binding and Counterpulsation in Cardiopulmonary Resuscitation

Charles F. Babbs, M.D., Ph.D.,* and William E. Blevins, D.V.M.**

*Associate Research Scholar, Biomedical Engineering Center, Purdue University, West Lafayette, Indiana, USA.

**Professor of Radiology, School of Veterinary Medicine, Purdue University, West Lafayette, Indiana, USA.

ABSTRACT

The history and potential of abdominal pressure as an adjunct to standard cardiopulmonary resuscitation (CPR) is reviewed. Abdominal pressure during CPR may be either static or phasic. Static abdominal pressure redirects limited blood flow generated by chest compressions toward the heart and brain, and away from the abdominal organs and lower extremities, thus boosting coronary perfusion pressure. Phasic abdominal pressure applied whenever chest pressure is released, in counterpoint to the rhythm of chest compression—so called interposed abdominal compression (IAC)-CPR—generates blood flow by an independent abdominal pump mechanism that augments the systemic perfusion created by chest compressions. New imaging data are presented in the form of cardiac angiograms during CPR in anesthetized dogs, illustrating the effects of interposed abdominal compressions added to otherwise standard CPR. The benefits vs. risks of IAC-CPR are discussed.

Key words: blood flow, CPR, IAC-CPR, interposed abdominal compression CPR, mechanisms, radiography, ventriculograms, x-ray

[Critical Care Clinics 1986; Vol 2(2): 319-332]

Dr. Babbs is supported by Research Career Development Award HL-00587 from the National Heart, Lung, and Blood Institute, U.S. Public Health Service.

INTRODUCTION

Three themes have dominated the history of abdominal compression during resuscitation from cardiac arrest. The first is the desirability of increasing peripheral resistance to non-vital organs, so that available blood flow can be directed toward the heart and the brain. The second is the possibility of augmenting venous return and preventing peripheral venous pooling by applying physical pressure of some sort to the lower portion of the body. The third is the danger that fatal hepatic trauma may occur if abdominal compression moves the liver beneath the sternum while chest compression is being applied. These considerations are the crucial factors in the benefit-risk equation for abdominal compression during cardiopulmonary resuscitation (CPR), and a considerable body of literature already exists that deals with their relative importance. Historically and practically, there have been two distinct approaches to abdominal pressurization during cardiac arrest and resuscitation. The first is that of sustained abdominal compression, which can be thought of as a noninvasive analog of cross-clamping of the aorta. The second is pulsed abdominal compression, which can be thought of as a non-invasive analog of intra-aortic (and intracaval) balloon pumping.

SUSTAINED ABDOMINAL COMPRESSION DURING CPR

Crile and Dolley, in 1906, noted the importance in cardiac resuscitation of securing a coronary artery pressure of at least 30 to 40 mm Hg, and advocated the use of a pneumatic pressure suit over the legs and abdomen, as well as epinephrine, to increase peripheral resistance and to prevent peripheral pooling of venous blood[12]. In 1914, Crile wrote in his book *Anemia and Resuscitation*: [22]

The author was able, personally, to produce on a recently deceased person, a total circulation which caused a pulse in the radial artery and bleeding of the peripheral vessels. Even a measurable blood pressure (registered by a sphygmomanometer) could be recorded through the combined effect of a tightly blown up rubber suit, which covered the lower extremities and the abdomen and the strong, rhythmical pressure through the hands, placed broadly on both sides of the thorax.

Shortly after the rediscovery of closed-chest cardiac massage by Kouwenhoven and colleagues in the late 1950s [18], Birch and coworkers studied dogs and baboons as animal models for CPR [6]. They produced ventricular fibrillation with transchest electric shock, and performed mechanical CPR with a pneumatic piston device. In one of the animals they exerted sustained abdominal pressure manually during chest compression, to "augment venous return." This animal at necropsy had liver rupture. A similar result was obtained in a second animal.

In 1967, Harris and associates studied a variety of interventions to improve closed-chest CPR, including continuous manual compression of the upper abdomen during CPR, in dogs with electrically induced ventricular fibrillation [15]. These studies were conducted with a Beck-Rand sternal compressor and fixed tidal volume ventilation (15 ml/kg) interposed after every fourth sternal compression. Carotid flows consistently increased by a factor of 1.5 to 2 when sustained

upper abdominal compression was added to otherwise conventional CPR in six dogs. However, they also found liver lacerations at necropsy in two of the six dogs, and so did not recommend sustained manual compression of the upper abdomen as an adjunct to CPR in clinical practice.

However, in 1971, Redding reinvestigated the benefit-risk of static abdominal binding in CPR in a study involving 145 dogs [25]. He demonstrated improved carotid artery flow and survival in experimental CPR with continuous abdominal compression by means of a blood pressure cuff secured around the mid-abdomen, and inflated to 150 to 200 mm Hg. Redding's model of cardiac arrest was produced by occlusion of the endotracheal tube for 6 to 9 minutes until blood pressure pulses ceased. Then, standard or experimental CPR was performed until pulsatile blood pressure returned or until 20 minutes had elapsed. Surviving animals were recovered from anesthesia and observed for 24 hours. Redding observed with absolute consistency that if aortic diastolic pressure during CPR could be raised above 40 mm Hg, pulsatile blood pressure could be restored. Standard CPR was effective in producing aortic diastolic pressure of 40 mm Hg or greater in only a few dogs, and in these animals spontaneous circulation returned. On the other hand, CPR augmented either by the drug methoxamine (a vasoconstrictor) or by abdominal binding usually led to aortic diastolic pressures above 40 mm Hg and to return of pulsatile blood pressure. Continuous abdominal binding in Redding's studies produced, on the average, 34 mm Hg greater aortic diastolic pressure than did standard CPR. In this study, Redding observed no greater incidence of liver damage during CPR with continuous abdominal binding than in similarly resuscitated animals without abdominal binding.

Redding's work is particularly important because the endpoint measured--survival--is the one of paramount clinical interest. In dogs receiving standard CPR for 20 minutes or less after asphyxic cardiac arrest, three of 15 animals resumed spontaneous circulation, and one of the 15 survived for 24 hours. However, in dogs receiving CPR with abdominal binding for 20 minutes or less after asphyxic cardiac arrest, 14 of 15 animals resumed spontaneous circulation, and nine survived for 24 hours. Subsequent studies with abdominal compression at 100, 150, and 200 mm Hg, and with the combination of abdominal compression and methoxamine, in 40 additional animals were consistent with these favorable results. These survival data reflect the effects of both increased benefits and increased risks of static abdominal binding during CPR, and quite clearly suggest that the benefits outweigh the risks.

More recently, Bircher and colleagues reported a preliminary study of experimental CPR in dogs in which a pediatric MAST suit was continuously inflated around the legs and abdomen for 10-minute periods, alternating with 10-minute control periods [7]. The results of this study were presented in September, 1979, and published in 1980. They found "no major complications attributable to the MAST, particularly no major lacerations of the liver" in 12 dogs receiving this treatment, which did increase diastolic arterial pressure and carotid artery flow nearly twofold. However, in a subsequent study, presented in June, 1980 [8], the authors repeated experiments with MAST trousers in another six dogs in order to observe the ease of restoration of spontaneous circulation and neurologic recovery thereafter. After 5 minutes of standard CPR, a pediatric MAST suit was inflated to 90 mm Hg and CPR was continued for 30 minutes. There was a doubling of carotid artery blood flow. However, there was also significant arterial hypoxemia and hypercarbia in this group compared with control animals receiving standard CPR, indicating gross intrapulmonic shunting in the MAST-CPR group. The authors attributed

blood gas deterioration to restriction of ventilation by the MAST suit, which was used in conjunction with the constant pressure ventilation system (a Michigan Instruments Thumper): Strikingly, all dogs in the MAST-CPR group had ruptured livers with hemorrhage into the peritoneal cavity, whereas there were no such injuries in seven dogs in the standard CPR group. In comparing their two studies, Bircher and coworkers concluded that "the mechanism of hepatic damage and hypoventilation may be the splinting of the liver immediately beneath the lower sternum."

In contrast, Chandra and Rudikoff and their coworkers at Johns Hopkins reported several CPR studies using sustained abdominal binding in animals and in humans that suggest that elevated blood pressures and flows can be achieved without liver damage [9, 27]. In a series of 15 dogs, they found that tightly binding the abdomen over an inflatable bladder with adhesive tape from the xyphoid to the iliac crest increased aortic systolic pressure from 28 to 57 mm Hg and carotid blood flow from 14 to 32 ml/min [27]. Carotid flow increased in 13 of the 15 dogs after abdominal binding, and there was no evidence of liver injury with mechanically controlled chest compression and abdominal binding in any of the 15 animals [27].

In Chandra's human studies [9], radial artery blood pressure was measured during brief (30-to 60-second) periods of abdominal binding in 10 patients experiencing cardiac arrest and standard CPR applied with a mechanical device. The abdomen was bound with an inflatable 30-cm square bladder that was positioned anteriorly on the patient's abdomen, extending from the lower ribs to the anterior superior iliac spine, held in place by a large strap, and inflated to approximately 100 mm Hg. Abdominal binding increased diastolic radial artery pressure from 35 to 50 mm Hg on the average. In six patients who underwent abdominal binding for longer periods (in excess of 4 minutes), higher arterial pressure persisted for the duration of abdominal binding. One of these six patients was successfully resuscitated, but died 24 hours later. None of the six patients showed evidence of abdominal visceral injury at autopsy. Specifically, no liver rupture was seen.

The issue of liver laceration is obviously crucial in any discussion of the overall benefit-risk of static abdominal compression in CPR. All published investigations report hemodynamic benefit of the procedure for several minutes or more. However, there is disagreement about the likelihood of liver damage and intra-peritoneal hemorrhage of such severity as to make successful resuscitation impossible. The weight of evidence, summarized in Table 1, is consistent with the hypothesis that liver rupture can be avoided with proper technique. One especially attractive means of securing the hemodynamic benefits of abdominal pressurization, while maintaining the low risk of liver trauma of standard CPR, is to compress the abdomen only during the diastolic phase of chest compression. This dynamic technique of interposed abdominal compression is under active investigation in several laboratories and clinical arenas and appears to offer a means of securing significant hemodynamic benefits without significant risk of liver trauma.

Table 1. Summary of Evidence Regarding Liver Laceration as a Result of Static Abdominal Pressure in CPR (SAC-CPR)

STUDIES SUGGESTING SAC-CPR DOES CAUSE LIVER RUPTURE			STUDIES SUGGESTING SAC-CPR DOES NOT CAUSE LIVER RUPTURE		
<i>Authors</i>	<i>No. of Subjects*</i>	<i>Technique</i>	<i>Authors</i>	<i>No. of Subjects*</i>	<i>Technique</i>
Birch et al. ⁶	2 dogs	Continuous manual pressure	Redding ²⁵	55 dogs	Blood pressure cuff around abdomen
Harris et al. ¹⁵	6 dogs	Continuous manual pressure (upper abdomen)	Rudikoff et al. ²⁷	15 dogs	Inflatable bladder + adhesive tape from xyphoid to iliac crest
Bircher et al. ⁸	6 dogs	Pediatric MAST suit	Bircher et al. ⁷	12 dogs	Pediatric MAST suit
			Chandra et al. ⁹	10 patients	Bladder + corset from ribs to iliac crest

*Number of dogs refers to animals subjected to abdominal pressurization, excluding control groups.

ABDOMINAL COUNTERPULSATION DURING CPR

Abdominal counterpulsation denotes the addition of interposed abdominal compressions to standard CPR (IAC-CPR). CPR is performed exactly as recommended in current American Heart Association standards [28], and in addition the abdomen is compressed alternately or reciprocally as chest compression is released. Zero or near-zero pressure is applied to the abdomen during chest compression, so that the liver is not splinted and trapped beneath the rib cage, as may occur with continuous abdominal compression or binding, but rather is free to move in response to trans-diaphragmatic pressure gradients. In the field or in the hospital, this technique of abdominal counterpulsation necessarily would require the services of an additional rescuer--a total of two or three rescuers, as illustrated in Figure 1.

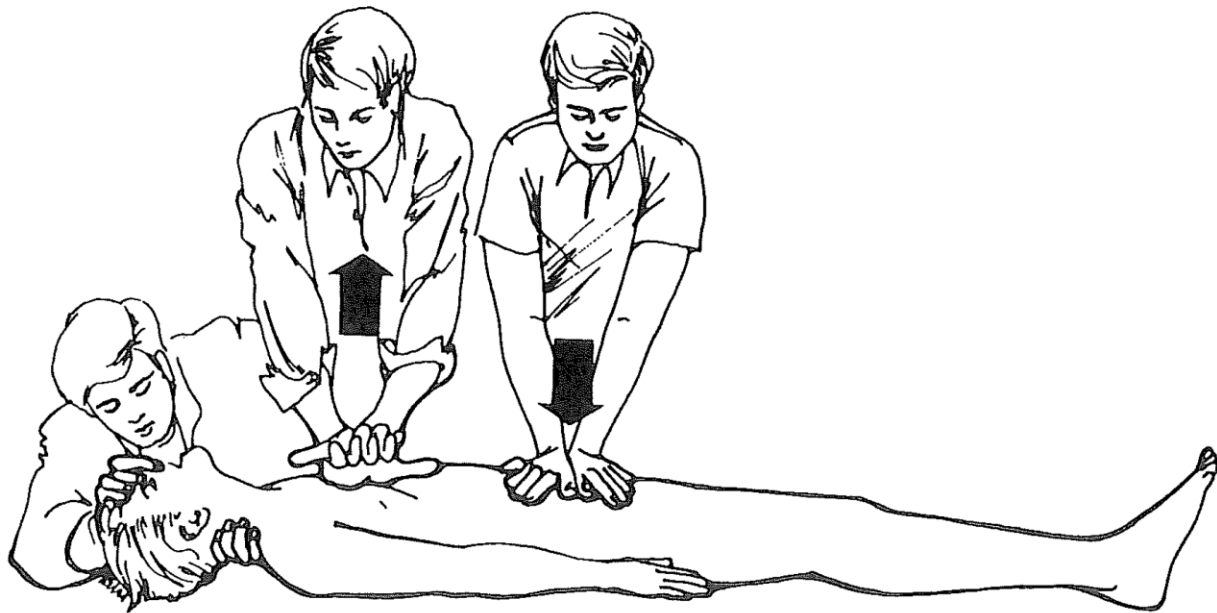


Figure 1. Artist's conception of basic rescuers performing abdominal counterpulsation (IAC-CPR). For clarity, the rescuers are shown on the same side of the victim.

The method of abdominal counterpulsation, or IAC-CPR, has been discovered independently by several research teams. The neglected report of Ohomoto and coworkers, at Tokyo Women's Medical College in 1976 [21], described an arrangement of two mechanical pistons, one that compressed the chest and another that compressed the abdomen. They termed the technique "countermassage" and found in preliminary studies that phased abdominal compression for 80% of the cycle time and chest compression for 25% of the cycle time appeared to improve carotid flow, mean aortic pressure, and short-term survival in anesthetized dogs with ventricular fibrillation. Rosborough and coworkers, in Houston, Texas [26], while attempting to develop an animal model of cough-CPR [13], combined simultaneous high-pressure lung inflation with abdominal compression. They found to their surprise that abdominal compression and ventilation alone could maintain carotid flow and aortic blood pressure during ventricular fibrillation in dogs, and they suggested the technique as a new CPR modality.

In our laboratory, the method was first demonstrated by Ralston serendipitously during a difficult resuscitation of a dog [24] (Fig. 2). In a subsequent controlled study, we compared blood pressures and flow during IAC-CPR with those generated by standard CPR in the same animal. Cardiac output during alternate 3-minute trials of the two techniques was measured in pentobarbital-anesthetized dogs during electrically induced ventricular fibrillation, using a modified indicator dilution technique adapted to the low-flow conditions of CPR [29].

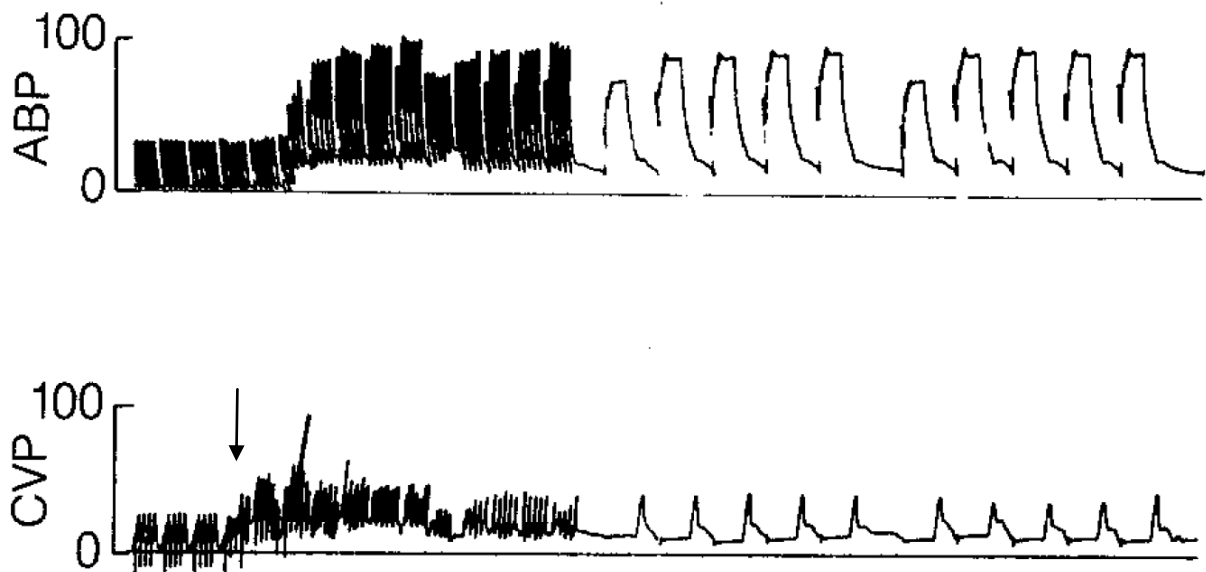


Figure 2. Original graphic record of brachial artery blood pressure (ABP, in mm Hg) and central venous blood pressure (CVP, in mm Hg) before and after onset of manual abdominal counterpulsation (arrow). The compression rate was 60 per minute; chart speed was increased on the right. Abdominal counterpulsation greatly improved systolic and diastolic arterial pressures, while CVP remained low. (Modified from Ralston, S. H., Babbs, C. F., and Niebauer, M. J.: Cardiopulmonary resuscitation with interposed abdominal counterpulsation in dogs. *Anesth. Analg.* 61:645-651, 1982.)

Ventricular fibrillation was induced electrically in 10 anesthetized dogs, and either IAC-CPR or standard CPR was initiated, while arterial and venous blood pressures and cardiac output were monitored. In this first study ventilation was provided by the controlled pressure ventilator of the Thumper. The two CPR methods were alternated every 3 minutes over a period of 30 minutes. IAC-CPR was performed by manual compression of the mid-abdomen with a standard 12-cm width blood pressure cuff, folded to rectangular dimensions of 12 x 15 cm and inflated with air to a thickness of 3 cm. The bladder of the cuff was attached to a transducer in order to monitor pressure applied to the abdomen.

During the release phase of chest compression, manual pressure was applied to the abdomen in such a way as to generate pressure pulses of 120 to 150 mm Hg. The position of the hands for abdominal compression was similar to that used in basic CPR for manual chest compression, except that the fingers were spread to provide a larger surface area of compression, approximately equal to that of the flattened blood pressure cuff. The duty cycle of abdominal compression was exactly complementary to that of chest compression-that is, 50% of the cycle

time. The addition of interposed abdominal compression to standard CPR significantly improved arterial pressures and perfusion. Figure 3 shows mean cardiac output and diastolic arteriovenous pressure differences for the animals in this study. The latter is particularly important, in our view, for promoting coronary flow and return of circulation after prolonged resuscitation.

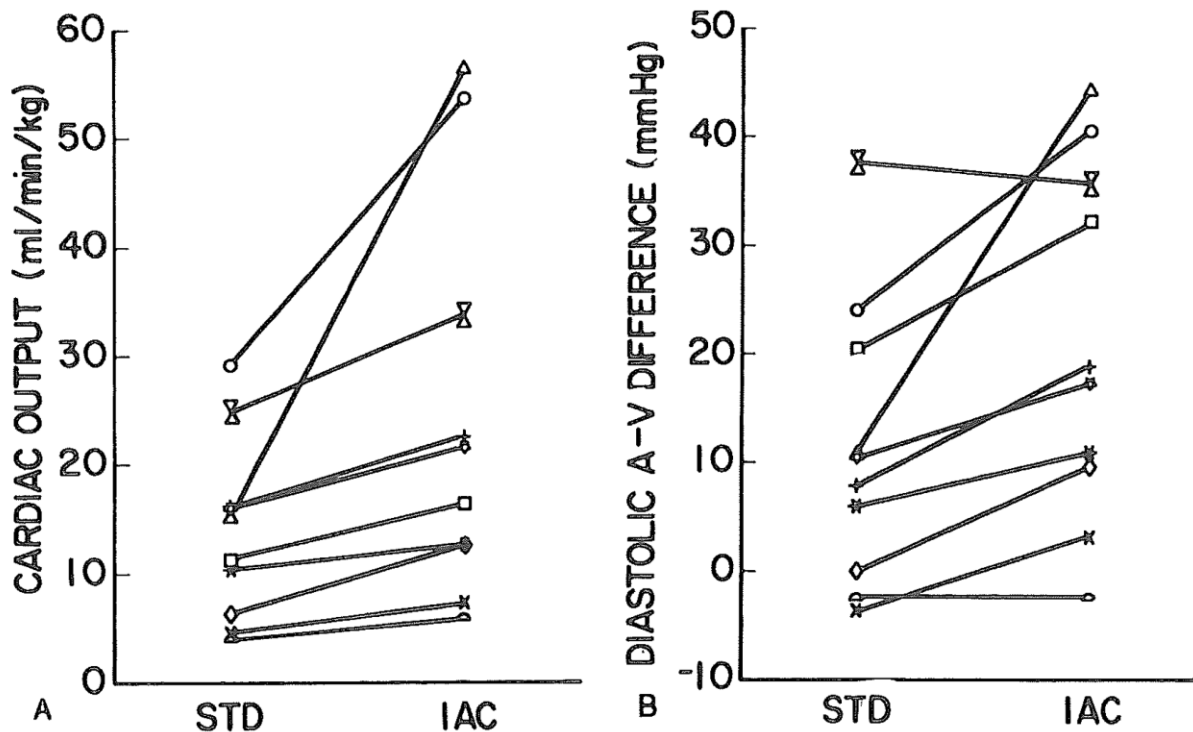


Figure 3. Paired observations of cardiac output (A) and central diastolic arteriovenous pressure difference (B) during standard CPR (STD) and CPR with interposed abdominal compressions (IAC) in 10 dogs. Each data point represents the mean of five trials in the same animal; each symbol represents one individual in the study.

Contrast angiocardigraphic studies in two animals confirmed improved hemodynamics with IAC-CPR compared with standard CPR. Figures 4A and 4B show spot films taken in the lateral projection during standard CPR in one animal at 4 and 13 seconds after injection of contrast medium into the left ventricle. Figures 4C and 4D show films taken at 4 and 13 seconds during IAC-CPR in the same animal. The films at 4 seconds show much greater regurgitation of contrast into the left atrium and pulmonary veins with standard CPR than with IAC-CPR. The 13-second films show faster clearance of contrast from the heart and aorta, suggesting greater blood flow.

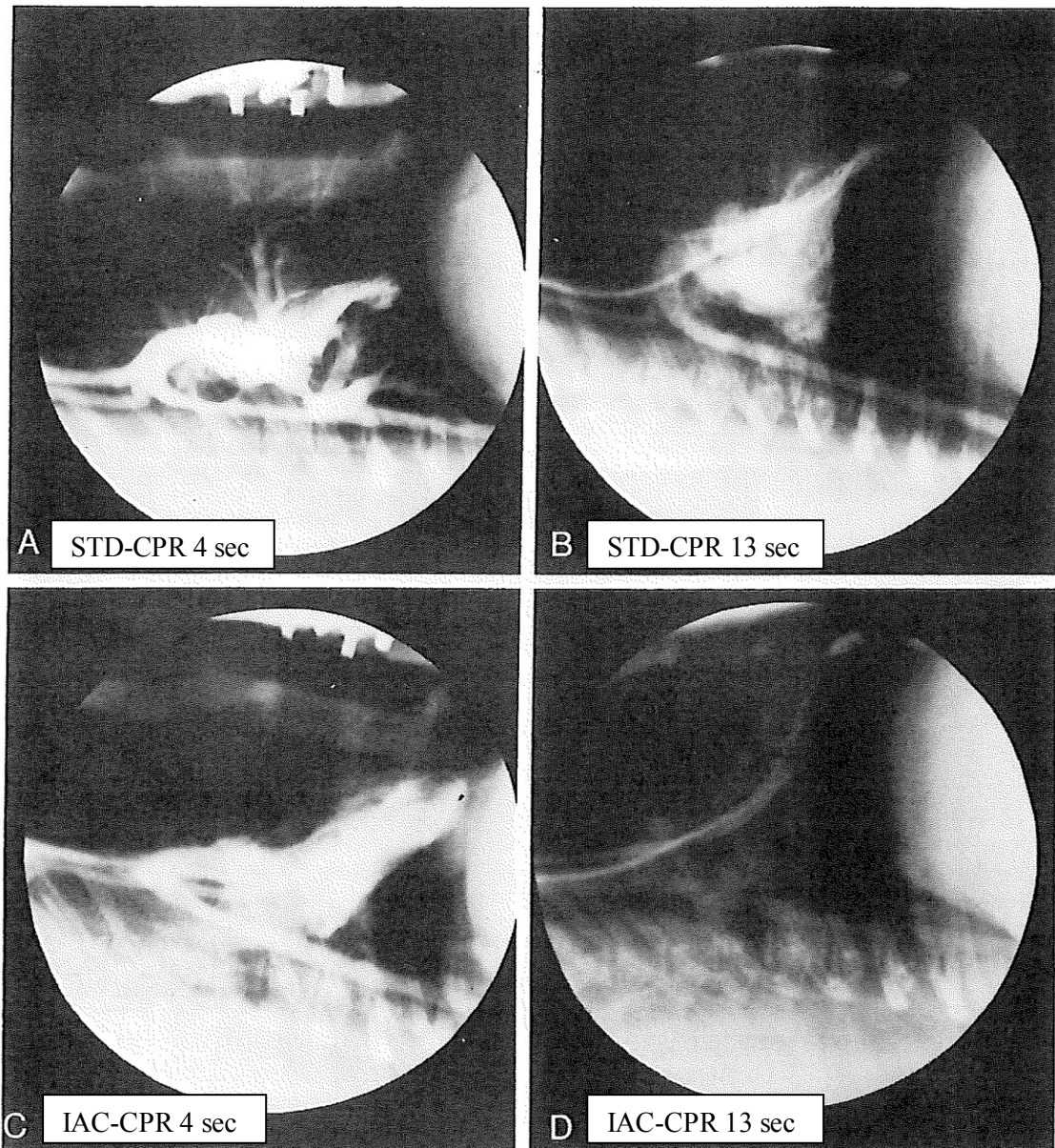


Figure 4. Spot films following injection of left ventricular contrast during standard CPR versus IAC-CPR in the same animal. Orientation: head left, tail right, sternum top. (A) Standard CPR 4 seconds after contrast injection. (B) Standard CPR 13 seconds after contrast injection. There is regurgitation of contrast into the left atrium and pulmonary veins, and poor washout after 13 seconds. (C) IAC-CPR 4 seconds after contrast injection. (D) IAC-CPR 13 seconds after contrast injection. There is improved opacification of the aorta, little backflow into the left atrium and pulmonary veins, and nearly complete washout after 13 seconds.

In a further study, we measured oxygen consumption in dogs with ventricular fibrillation, supported by either standard CPR or IAC-CPR. Oxygen delivery during CPR was measured by a spirometer in a closed system for positive pressure ventilation, slaved to the timing circuit of a Thumper mechanical chest compressor. Gas withdrawn from a spirometer bell into a bellows was used to ventilate the animal with a constant tidal volume. In effect, therefore, this study was performed with a volume-controlled ventilator. Special valves were included in the circuit to protect the spirometer bell from positive pressure during lung inflation. Ventricular fibrillation was induced electrically in 10 anesthetized dogs, and either IAC-CPR or standard CPR was initiated, while arterial and right ventricular blood pressures and oxygen consumption were monitored. The two CPR methods were alternated every 3 minutes over a period of 30 minutes.

Figure 5 shows a graphic record from an animal in this series. The two top tracings indicate spirometer volume. The second channel is the spirometer volume electronically filtered to damp out oscillations. The overall slopes of the upper two channels indicate oxygen uptake. After abdominal counterpulsation was added to standard CPR, brachial artery pressure and oxygen uptake improved significantly.

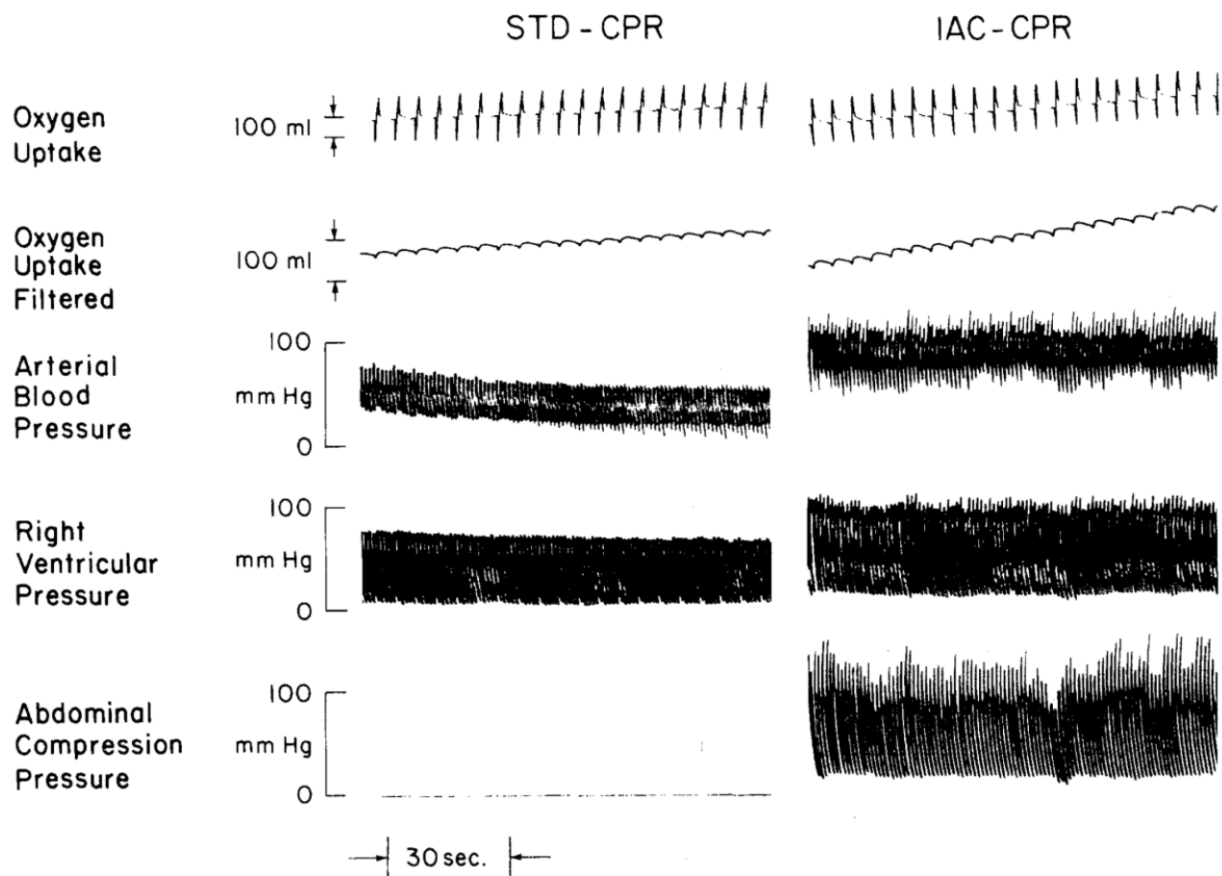


Figure 5. Graphic records of oxygen uptake and blood pressures during experimental CPR in a dog. The recording of IAC-CPR (right) was inscribed less than 1 minute after that of standard CPR (left). Oxygen uptake and diastolic arterial pressure are clearly improved by abdominal counterpulsation.

At about the same time, Coletti and coworkers in New York were studying the influence of abdominal counterpulsation on cerebral and coronary blood flow in a canine model of cardiogenic shock [10, 11]. They found that when manual abdominal compressions were interposed between heartbeats, as judged from the electrocardiograph trace, both cerebral and coronary perfusion, measured with implanted electromagnetic flow meters, increased. Walker and co-workers in Detroit studied perfusion of the cerebral cortex during IAC-CPR versus standard CPR in a canine model [31]. They used both manual chest and manual abdominal compression in large supine dogs in which cardiac arrest was produced by intravenous potassium chloride. The chest compression was done in a side-to-side manner, as is accepted practice in veterinary medicine [22]. They found that cerebral cortical blood flow, measured with a thermal washout method [16] immediately after beginning CPR, averaged 0.06 ml/min/g in animals receiving chest compression alone and 0.27 ml/min/g in animals receiving chest compressions with IAC. Voorhees and associates, using the radioactive microsphere technique in dogs with ventricular fibrillation, found cerebral perfusion of 0.28 ml/min/g with IAC-CPR [30], a value significantly greater than that during standard CPR and nearly identical to that reported by Walker and colleagues. These values obtained with IAC-CPR represent approximately 30% to 50% of pre-arrest cerebral perfusion in dogs under anesthesia.

These animal studies suggesting the value of interposed abdominal compressions as an adjunct to CPR have been confirmed in an electrical model of the circulation by Babbs and coworkers [2, 3]. We modeled the heart and blood vessels as resistive-capacitive networks, pressures as voltages, blood flow as electrical current, blood inertia as inductance, and the cardiac and venous valves as diodes. Pressurization of the chest and abdomen, as would occur in CPR, was simulated by application of half-sinusoidal voltage pulses, between vascular capacitances and ground. The model was typically arranged to simulate thoracic pump CPR [1], in which blood is impelled by compression of all intrathoracic vascular structures. Interposed abdominal compressions added to chest compression in this model produced flow augmentation, according to the expression:

$$\text{flow} = \alpha P_{th} + \beta P_a ,$$

where P_{th} = peak intrathoracic pressure, P_a = peak abdominal pressure, and α and β are constants ($\alpha > \beta$). Interposed abdominal compressions enhanced simulated blood flow to the heart and brain, as well as total flow, in the electrical model. The degree of flow augmentation depended on both the abdominal pressure applied and the peripheral vascular resistance. Simulated IAC-CPR with high peripheral resistance values in this model generated current flow to the head and neck corresponding to a blood flow roughly 70% of normal, the highest value obtained in any simulation [2].

Stimulated by these encouraging laboratory findings, clinical studies of IAC-CPR are now under way at several centers. Berryman and Phillips have reported improved mean arterial pressure during periods of abdominal counterpulsation in patients resuscitated in the emergency department [5]. Howard and coworkers have found that the addition of interposed abdominal compressions to otherwise standard CPR significantly improved arteriovenous pressures in a

small population of 12 patients with witnessed cardiac arrest who were promptly brought to the hospital [17]. Stueven and coworkers in Milwaukee, however, found that brief periods of IAC-CPR applied by paramedics in the field to patients unresponsive to initial defibrillation did not alter resuscitation success [19]. No study has yet shown that long-term survival is improved by IAC-CPR or that neurologic function among survivors is as good as or better than with standard CPR. Such evidence will probably be needed before IAC-CPR can be integrated into basic life-support protocols taught to lay persons and implemented on a national scale.

Regarding the safety of abdominal counterpulsation in CPR, liver laceration was not observed as a complication in the animal studies of Voorhees and associates [29, 30]. Hence, the trials of IAC-CPR in animals that also received trials of standard CPR must not have caused hepatic trauma. Likewise, in the clinical studies of Berryman, Howard, Mateer, and their coworkers [5, 17, 19] trauma to abdominal organs as a result of interposed abdominal compressions has not been reported.

Abdominal counterpulsation during CPR has not caused esophageal regurgitation in animal studies. Clinical studies to date have not found the incidence of regurgitation during abdominal counterpulsation to be significantly greater than that during standard CPR. Nonetheless, it is important to discuss the possibility of provoking regurgitation and subsequent aspiration by IAC-CPR. In our animals, a tracheal tube was securely in place, and gastric insufflation during ventilation did not occur. Gastric distention is a common sequela of mouth-to-mouth ventilation in humans [28], and it is possible that abdominal pressure may induce vomiting after the stomach is distended with air. However, one may speculate that if the IAC-CPR technique were used consistently from the beginning of resuscitation, gastric distention might be entirely prevented by the abdominal counterpressure.

We have recently studied this possibility in the laboratory [4]. A canine model was employed in which a common ventilation pressure was applied to separate cuffed esophageal and tracheal tubes. Gas entering the stomach was collected via a preplaced gastrostomy tube leading to a bell spirometer. Gas entering the lungs was measured with a Wright Respirometer in series with the endotracheal tube. During standard CPR, measurable gastric gas volume was collected in 28 of 30 trials, with a mean value of 215 ml per ventilation. During IAC-CPR, in which abdominal pressure was maintained during ventilation after every fifth chest compression, measurable gastric gas was collected in only 15 of 30 trials, with a mean value of 40 ml per ventilation. These results support the hypothesis that abdominal counterpulsation performed from the outset of resuscitation tends to reduce the incidence of gastric insufflation and attendant complications. The most probable situation in which interposed abdominal compressions might induce regurgitation would be if the technique were added after a period of conventional CPR, as might occur after others come to the aid of a lone rescuer.

SUMMARY AND CONCLUSIONS

During the past 15 years, many different studies have documented improved blood pressure and blood flow above the diaphragm when some type of abdominal compression was added to conventional CPR, either in animals or in humans. Rhythmically interposed abdominal compressions seem to provide even greater hemodynamic benefit than continuous abdominal binding. Both total flow and the distribution of flow to vital organs above the diaphragm are improved, while the chances of liver entrapment and damage during chest compression are reduced.

The technique of interposed abdominal compression can be performed with the bare hands of a second or third rescuer. It requires no special equipment, and could be easily incorporated into existing training programs for basic rescuers. In this sense the technique may constitute a logical evolution in basic life support, if subsequent clinical research confirms that it improves outcome.

REFERENCES

1. Babbs, C. F.: New versus old theories of blood flow during cardiopulmonary resuscitation. *Crit. Care Med.*, 8:191-195, 1980.
2. Babbs, C. F., Ralston, S. H., and Geddes, L. A., Theoretical advantages of abdominal counterpulsation in CPR as demonstrated in a simple electrical model of the circulation. *Ann. Emerg. Med.*, 13:660-671, 1984.
3. Babbs, C. F., Weaver, J. C., Ralston, S. H., et al.: Cardiac, thoracic, and abdominal pump mechanisms in CPR: Studies in an electrical model of the circulation. *Am. J. Emerg. Med.*, 2:299-308, 1984.
4. Babbs, C. F., Schoenlein, W. E., and Lowe, M. W.: Gastric insufflation during IAC-CPR compared to standard CPR in a canine model. *Am. J. Emerg. Med.*, 3:99-103, 1985.
5. Berryman, C. R., and Phillips, G. M.: Interposed abdominal compression-CPR in human subjects. *Ann. Emerg. Med.*, 13:226-229, 1984.
6. Birch, L. H., Kenney, L. J., Doornbos, F., et al.: A study of external cardiac compression. *Mich. Med.*, 61(11):1346-1352, 1962.
7. Bircher, N., Safar, P., and Stewart, R.: A comparison of standard, MAST-augmented, and open-chest CPR in dogs--A preliminary investigation. *Crit. Care Med.*, 8:147-152, 1980.
8. Bircher, N. G., Alifimoff, J. K., and Safar, P.: Perfusion augmentation during cardiopulmonary resuscitation (CPR). *Circ. Shock*, 7:210, 1980.

9. Chandra, N., Snyder, L. D., and Weisfeldt, M. L.: Abdominal binding during cardiopulmonary resuscitation in man. *J.A.M.A.*, 246:351-353, 1981.
10. Coletti, R. H., Kaskel, P. S., Cohen, S. R., et al.: Abdominal counterpulsation (AC)--A new concept in circulatory assistance. *Trans. Am. Soc. Artif. Intern. Organs*, 28:563- 566, 1982.
11. Coletti, R. H., Kaskel, P. S., and Bregman, D.: Abdominal counterpulsation (AC)--Effects on canine coronary and carotid blood flow. *Circulation*, 66(II):135, 1982.
12. Crile, G., and Dolley, D. T.: Experimental research into resuscitation of dogs killed by anesthetics and asphyxia. *J. Exp. Med.*, 8:713-720, 1906.
13. Criley, J. M., Blaufuss, A. H., and Kissel, G. L.: Cough-induced cardiac compression--A self-administered form of cardiopulmonary resuscitation. *J.A.M.A.*, 236(11):1246-1250, 1976.
14. Geddes, L. A., and Babbs, C. F.: A new technique for the measurement of cardiac output during cardiopulmonary resuscitation. *Crit. Care Med.*, 8:131-133, 1980.
15. Harris, L. C., Kirimli, B., and Safar, P.: Augmentation of artificial circulation during cardiopulmonary resuscitation. *Anesthesiology*, 28(4):730-734, 1967.
16. Hoehner, P. J., Krause, G. S., White, B. C., et al.: Determination of cerebral cortical blood flow: A thermal technique. *Ann. Emerg. Med.*, 12:2-7, 1983.
17. Howard, M., Carrubba, C., Guinness, M., et al.: Interposed abdominal compression CPR: Its effects on coronary perfusion pressure in human subjects. *Ann. Emerg. Med.*, 13:989-990, 1984.
18. Kouwenhoven, W. B., Jude, J. R., and Knickerbocker, G. G.: Closed-chest cardiac massage. *J.A.M.A.*, 173:1064-1067, 1960.
19. Mateer, J. R., Stueven, H. A., Thompson, B. M., et al.: Preliminary results: Interposed abdominal compression CPR vs. standard CPR in prehospital cardiopulmonary arrest. *Am. J. Emerg. Med.*, 2:354, 1984.
20. Nagel, E. L., Fine, E. G., Krischer, J. P., et al.: Complications of CPR. *Crit. Care Med.*, 9:424, 1981.
21. Ohomoto, T., Miura, I., and Konno, S.: A new method of external cardiac massage to improve diastolic augmentation and prolong survival time. *Ann. Thorac. Surg.*, 21:284-290, 1976.

22. Overbeck, W.: The past: Historical views concerning cardiac arrest and resuscitation. In Stephenson, H. E. (ed.): Cardiac Arrest and Resuscitation. Edition 4. St. Louis, C. V. Mosby, 1974.
23. Palich, W. E., and Gordon, A. S.: Cardiopulmonary resuscitation of dogs: Principles and practice. J. Am. Vet. Med. Assoc., 151:1719-1732, 1967.
24. Ralston, S. H., Babbs, C. F., and Niebauer, M. J.: Cardiopulmonary resuscitation with interposed abdominal compression in dogs. Anesth. Analg., 61:645-651, 1982.
25. Redding, J. S.: Abdominal compression in cardiopulmonary resuscitation. Anesth. Analg., 50:668-675, 1971.
26. Rosborough, J. P., Niemann, J. T., Criley, J. M., et al.: Lower abdominal compression with synchronized ventilation--A CPR modality. Circulation, 64(IV):303, 1981.
27. Rudikoff, M. T., Maughan, W. L., Effron, M., et al.: Mechanisms of blood flow during cardiopulmonary resuscitation. Circulation, 61:345-352, 1980.
28. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). J.A.M.A., 244:453-509, 1980.
29. Voorhees, W. D., Babbs, C. F., and Niebauer, M. J.: Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. Ann. Emerg. Med., 12:128-135, 1983.
30. Voorhees, W. D., Ralston, S. H., and Babbs, C. F.: Regional blood flow during cardiopulmonary resuscitation with abdominal counterpulsation in dogs. Am. J. Emerg. Med., 2:123-128, 1984.
31. Walker, J. W., Bruestle, J. C., White, B. C., et al.: Perfusion of the cerebral cortex using abdominal counterpulsation during CPR. Am. J. Emerg. Med., 2:391-393, 1984.